## Modelling the evolutionary dynamics of nutrient-deprived cancer cells using phenotype-structured differential equations.

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Glucose and oxygen are primary energy sources for cancer cells. Several lines of evidence support the idea that changes in gene expression levels (e.g. MCT1, HIF1) elicit metabolic reprogramming of cancer cells in nutrient-poor environments, promoting cancer cell survival and disease progression. A more in-depth theoretical understanding of the evolutionary processes at the root of cancer cell adaptation to nutrient deprivation can be achieved through analysis and numerical simulation of structured-population models. The focus of this talk is on non-local partial differential equations modelling of the adaptive dynamics of a population of cancer cells structured by the level of gene expression [1, 2]. First, I will present an experimentally-informed mathematical model of a well-mixed population, which was calibrated with data from in vitro experiments on glucose-deprived aggressive cancer cells. Then, I will present spatially explicit extensions of the modelling framework that shed light on the evolutionary processes responsible for the emergence of intratumour phenotypic heterogeneity in vascularised tumours.

## References

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